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The Bristol Medico-Chirurgical Journal

"Scire est nescire, nisi id me
Scire alius sciret."

SUMMER, 1929.

FOREIGN BODIES IN THE GULLET.

BY

E. WATSON-WILLIAMS, M.C., Ch.M., F.R.C.S.,
Surgeon-in-charge Ear, Nose and Throat Department, Bristol Royal Infirmary.

THE treatment of patients with foreign bodies impacted in the gullet is a branch of surgery that has in recent years undergone a complete change. Although the œsophagus and stomach were examined in the living subject by means of a tube as far back as the middle of the eighteenth century, clinical application of this method has been developed only during the twentieth. Before that development the means of dealing with such accidents were distinctly primitive. The obstructing mass was pushed down or hooked up by a blind instrumentation. If this failed, an external œsophagotomy in the neck was necessary. Both procedures were fraught with considerable risk, and

accompanied by a serious mortality. One must admire the ingenuity of the expanding probang and the hinged coin-catcher. But in so delicate a tube as the œsophagus the use of such instruments, gentle and skilful though it were, carried a real danger of damage; not solely from the instrument itself, but still more from the sharp edges or points of the foreign body. In a civilized community these appliances have now only one place—the museum.

The cardinal principle of treatment is the avoidance of all blind methods, every step being conducted under direct vision. The patient or parent generally knows what the foreign body is, and how long it has been present. Often, too, we can get some idea of where it is lodged. It must, however, be remembered that babies swallow a strange assortment of objects, and the possibility of a foreign body should not be overlooked in the case of a small patient who has suddenly developed dysphagia. The actual degree of this symptom varies much. A smooth, flat, foreign body may cause little disturbance; more commonly fluids only can be taken. In a few cases nothing at all can be swallowed, owing to the size of the foreign body or to reflex spasm; while if the obstruction is low down food is taken but immediately returned. Difficulty in swallowing is not always due to the actual presence of a foreign body. Damage inflicted by its passage, or during attempts to dislodge it, may from spasm increase the "feeling that there is something there."

Pain indicates irritation or actual laceration of the œsophagus. With slight damage it occurs only on swallowing, and fluids can usually be taken. I have records of six cases of this nature in which the laceration was verified by œsophagoscopy. In two cases it had

been caused by a probang, in one by thrusting down a sweet (? sharp edge or finger-nail), in three by the passage of the foreign body itself. For example, a woman who said she had swallowed a piece of glass in preserved fruit was found to have a deep laceration of the mucosa extending 2 cm. down from the lower border of the cricoid. In such cases a disinfectant mouthwash is used at frequent intervals, and formalin lozenges can be sucked. Food should be fluid, sterile and iced, and if necessary half a drachm of orthoform powder may be given twenty minutes before feeding. Bismuth should follow a meal:—

℞. Bismuth. Oxycarb. .. gr. xx.
Lactos. .. .. .. gr. xl.

A powder to be placed on the tongue and swallowed dry.

Where more serious damage has been inflicted there may be complete inability to swallow, attempts increasing the pain which does not cease in the intervals. Some fever is usual, and in a bad case the breath is fetid. Reflex salivation adds materially to the discomfort, and these patients should be nursed prone, the head to one side with only a small pillow under the occiput. In this position saliva can dribble from the mouth, instead of overflowing into the trachea and rendering rest impossible. Proctoclysis may be needed for two or three days until a fluid diet is possible. Three of my patients have needed this treatment. A still more serious condition was found in another, who said she had swallowed a plum-stone. A probang had been passed, without retrieving it, but from that time she could swallow nothing. I found that immediately below the œsophageal entrance the mucosa had been stripped from almost the whole

circumference over a distance of just over 2 cm., and hung in the lumen like an œdematous polypus. Gastrostomy was performed: after three weeks she was able to take fluids and made a complete recovery.

In none of the above cases was any foreign body seen. Two further cases are described below. In one a safety-pin penetrated the wall of the hypopharynx; the patient recovered. In the second a sharp splinter of bone had been made to pierce the œsophagus, with fatal results.

At the other end of the scale are those patients whose only evidence of swallowing is the sudden disappearance of an object held in the mouth, a pin or denture. It is curious how often a missing denture is found in the grate! A skiagram should always immediately precede any operative search for the foreign body—immediately, as these bodies sometimes change their position. Before resorting to this we have, of course, examined the pharynx and larynx with the aid of a mirror. Quite often this method suffices to locate an object that the patient thought farther down; and we must not forget that a body in the neighbourhood of the Eustachian orifice may be "felt" along the border of the thyroid cartilage. A skiagram will certainly show a denture, a needle, or any but the most minute fragment of metal. Figures 41 and 42 show the appearance of a coin and a toy watch. Glass, celluloid, fish-bones, meat and possibly small pieces of wood or bone may escape detection, though even with these an opaque meal may afford some help. One can recall the case of a small baby that was subjected to œsophagoscopy because he had "swallowed" a safety-pin, which a subsequent skiagram showed in the nose! In a very similar case I was myself invited to remove from the

gullet of a baby three weeks old a safety-pin that he had "swallowed." The skiagram revealed it in the naso-pharynx, where it could not be seen from the mouth and was felt only with considerable difficulty. Once located its removal was easy. (Figures 37 and 43.) On another occasion I was asked to retrieve by means of the œsophagoscope a needle that the skiagram disclosed in the cæcum.

When we have thus located the foreign body, or when we feel, despite negative skiagraphy, that it is reasonably probable that there is one in the gullet, we proceed to examine this region by means of an œsophagoscope. The latter is a metal tube of size appropriate to the age of the patient, with a small electric light at the distal end. I use it without a handle, as being more delicate in manœuvre. It is passed down with great gentleness, no force at all being permissible; and the œsophagus is closely examined during the whole movement, to avoid "over-riding." The need for care cannot be over-emphasized; even an object so blunt as a farthing has been made to penetrate the thin wall of the œsophagus. I prefer a general anæsthetic for this operation, except with nurslings. It is necessary with children, and few adults even can tolerate the presence of the tube for many minutes. It is extremely important that everything should proceed smoothly and without hurry or fuss. Coins are, as a rule, easily removed, though even with these one may regret "demonstrating" instead of removing the coin as soon as found. The appearance of a half-penny when viewed through the œsophagoscope is shown in Figure 22. With bulky objects, needing to be divided with shears, or bodies having hooks or spikes that require disimpaction, a problem of real difficulty may occur. Finally, special

instruments may be called for, such as the "closer for safety-pin point upward," illustrated in Figure 45.

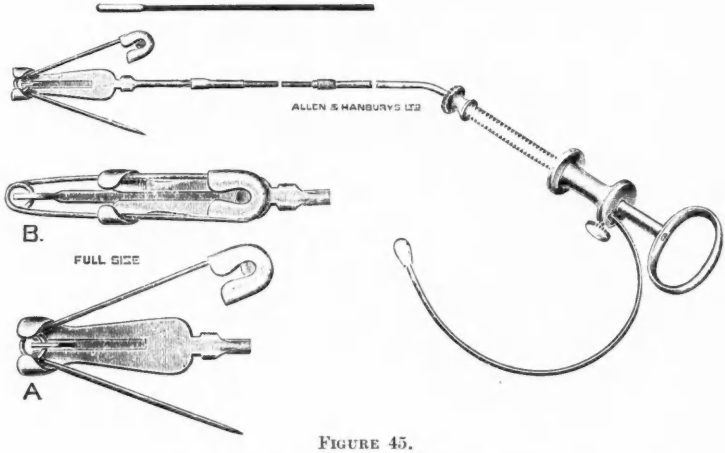


FIGURE 45.
Safety-pin closer, showing method of use.

A short description is appended of the fifty-three objects in this series, so far as it is interesting (or recorded). Measurements are in each case from the incisor teeth to the nearest part of the foreign body. The youngest patient was three weeks old, the oldest 73 years, and with the one exception mentioned all made a good recovery.

Coins. Twenty-four Cases.

Sixpence.

Age 1. Coin three days in throat, can take fluids only. Coin found at 10 cm. from teeth and removed. (Fig. 19.)

Penny. Two cases.

Age 11. A choir-boy, holding a penny for the collection in his hand put the latter to his mouth to cover a cough, and the coin slipped down. Recovered from the thoracic inlet. (Fig. 2.)

Age 6. A penny at level of top of sternum removed. (Fig. 3.)

PLATE X.

FIG. 1

FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIG. 6

FIG. 7

FIG. 8

FIG. 9

FIG. 10

FIG. 11

FIG. 12

FIG. 13

FIG. 14

FIG. 15

FIG. 16

FIG. 17

FIG. 18

FIG. 19

FIG. 20

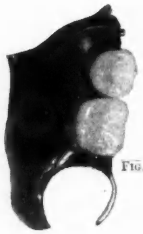


FIG. 21

FIG. 22

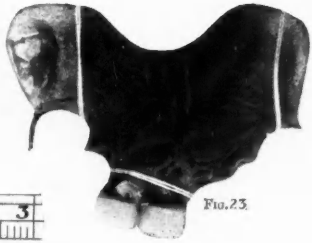
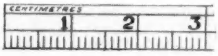


FIG. 23



Half-penny. Eighteen cases.

Age 1. Coin present two days, no symptoms. Radiogram shows it level with top of clavicle ; removed. (Fig. 1.)

Age 2. Coin swallowed same day, removed from level of suprasternal notch. (Fig. 4.)

Age 3. Coin just below cricoid, removed in 1 minute 40 seconds. (Fig. 5.)

Age 4. Coin at thoracic inlet ; removed.

Age 3. Coin at thoracic inlet ; removed. (Fig. 41.)

Age 4. Coin in centre of chest, found at 20·5 cm. from teeth ; removed. (Fig. 6.)

Age 4. Coin present 3 days, can take no solids. Skiagram shows it behind heart. Removed from behind left auricle at 25·5 cm. in 15 minutes. (Fig. 7.)

Age 7. Removed from thoracic inlet.

Age 3. Removed from just above sternum. (Fig. 8.)

Age 3. No symptoms. Coin swallowed same day, found behind cricoid and removed. (Fig. 9.)

Age 2. Top of coin level with bottom of cricoid ; removed.

Age 5. Coin removed from just below cricoid in 1 minute 30 seconds. (Fig. 10.)

Age 3. Removed from just above sternum. (Fig. 11.)

Age 4. Found at 12 cm. from teeth, removed in 1 minute 15 seconds. (Fig. 12.)

Age 6. Coin at 15 cm. from teeth, *i.e.* well in thoracic inlet. Removed in 2 minutes 12 seconds. (Fig. 13.)

Age 4. This coin I found 1 cm. below cricoid, *i.e.* at thoracic inlet. While demonstrating it, it was dislodged, and slipped down. It was recovered from just above the cardia ; time 16 minutes. (Fig. 14.)

Age 3. Coin two days in throat, no symptoms ; removed from level of thoracic inlet. (Fig. 15.)

Age 3. Removed from just above sternum. (Fig. 16.)

Farthings. Three cases.

Age 2. Coin swallowed two days before, can take fluids only; some dyspnœa. Coin removed from level of clavicle. (Fig. 17.)

Age 4. "He has swallowed a half-penny." A farthing was found at level of left bronchus, 5 cm. below thoracic inlet. Time 20 minutes. (Fig. 18.)

Age 4. "Was playing with his money-box and swallowed a farthing." Skiagram shows it level with top of sternum. On passing œsophagoscope edge of coin was seen and demonstrated—it showed particularly well. On removal it was found that forceps had grasped two exactly apposed farthings. (Fig. 20.)

Bones. Ten Cases.

Female, aged 43. A chicken bone swallowed the day before, removed from 35 cm. below the teeth. (Fig. 24.)

Female, aged 42. A rabbit bone lay in the left pyriform fossa, the point just visible by indirect laryngoscopy. (Fig. 25.)

Male, aged 33. Meat bone swallowed three days before; œdema of left pyriform fossa observed by indirect laryngoscopy. Bone removed from immediately beneath this point by direct œsophagoscopy. (Fig. 26.)

Female, aged 38. A rabbit bone lying behind the epiglottis, the sharp points causing much distress as they pricked the opposite walls of the pharynx every time the patient retched, which was frequently. (Fig. 27.)

Male, aged 11. This case is the tragedy of the series. While eating rabbit a bone stuck in his throat. His mother thrust her finger down and "cleared the throat." Next day he was brought to me looking very ill, unable to swallow at all, with great pain low in the neck, worse on attempting to swallow. The breath was fetid and the temperature 100·4° F. A radiogram failed to show any foreign body, but does show some commencing opacity of the tissues behind the top of the sternum (not noted before operation). I passed an œsophagoscope, and found immediately below the cricoideus a sharp splinter

PLATE XI.

FIG.24

FIG.31

FIG.28

FIG.25

FIG.32

FIG.29

FIG.26

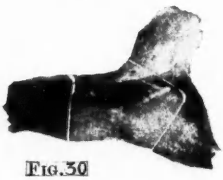


FIG.30

FIG.33

FIG.34

FIG.27

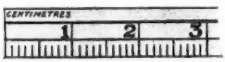


FIG.35

FIG.36

FIG.37

FIG.38

FIG.39

FIG.40

(NOTE—Fig. 28 not to scale).

of bone (Fig. 28) sticking out of the left wall of the gullet, which was œdematous. The bone, which measured 2·2 cm., had penetrated the wall for about one-third of its length. On removing it, beads of pus began to escape. With punch forceps I opened an abscess into the lumen, and about a drachm of stinking pus welled up. Next day he could swallow fluids, but seemed ill, and on the third day there was definite broncho-pneumonia. An area of dullness could be percussed rather to the right of the mid-line in first and second intercostal spaces in front. The radiogram (Fig. 44) showed a large patch of opacity. He remained very ill, so ill that no further operation could be done, and had attacks of faintness followed by gulping up of fetid pus—the abscess was draining, though not properly. Subsequent radiograms showed a diminution of the shadow, but increasing lung changes. Getting steadily worse, he died on the seventh day from the accident. At autopsy we found an abscess cavity, fairly well defined, running down outside the left wall of the œsophagus, and ending among the great vessels, and severe broncho-pneumonia. Almost certainly the bone had been pushed through the œsophageal wall by well meaning but disastrous attempts to dislodge it. It is noteworthy that splinters of bone from soups and stews, that might be supposed to be sterile, always cause severe and immediate infection if they penetrate; while pins and toys that are often not even reasonably clean tend to produce much less damage.

Female, aged 42. A mutton bone 17 cm. from the teeth, *i.e.* in thoracic inlet. (Fig. 29.)

The large bone in Figure 30 is included here to show what people will sometimes attempt to swallow; it appears to have been lodged behind the cricoid, and was hawked up by the patient while preparations were being made to remove it.

Female, aged 54. A large fish bone, apparently from skull of haddock, at thoracic inlet. (Fig. 31.)

Male, aged 50. Fish bone embedded in the vallecula. (Fig. 32.)

Male, aged 33. Fish bone in base of tonsil, out of sight. (Fig. 34.) The difficulty in these two cases was more that of detecting the almost hair-like foreign body than of removing it.

Male, aged 28. Stout fish bone at thoracic inlet. (Fig. 33.)

Meat. Seven Cases.

Pieces of meat may be insufficiently chewed and so become impacted on swallowing being attempted; or there may be some pathological condition of the œsophagus producing arrest of a normal bolus. The cases included here are those only in which the patient came "because something stuck."

Female, aged 59. A piece of meat stuck the day before; a probang had been used, which pushed it down about an inch. The meat was firmly held by the lower part of the inferior constrictor, and on removal was found to be two inches long, the middle constricted as if a string had been tied round it.

Female, aged 54. Bolus of hard gristly meat impacted at œsophageal entrance.

Female, aged 48. Some meat had stuck the previous day, since when "everything she swallows comes up at once"; pain behind the lower end of sternum. She has "always had a small swallow." A large piece of meat was found firmly impacted in the cardia; so firmly that many fragments broke off when removal was attempted and the whole operation took nearly an hour.

Male, aged 73. Never any trouble in swallowing till a week ago some meat stuck. Since then he has been unable to swallow anything at all. The skiagram shows an apparently normal œsophagus with barium arrested in centre. At 19 cm. from teeth meat was found, occupying 7 cm. of the lumen. On removal, at 25 cm. was seen a small ulcer of the posterior wall, bleeding easily on touching. The lumen beyond was quite normal. Pathological report: carcinoma.

Female, aged 55. Meat stuck in throat one day. A bolus of meat was found just above the cardia, which was apparently

PLATE XII.

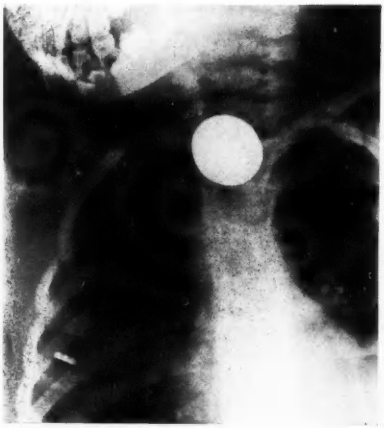


FIG. 41

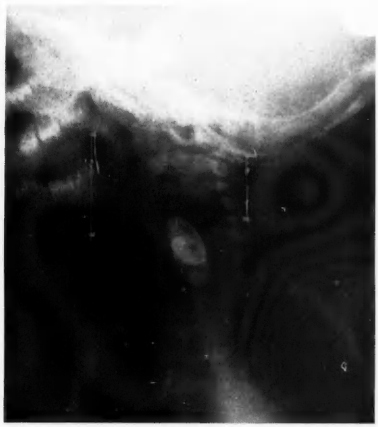


FIG. 42

FIG. 43

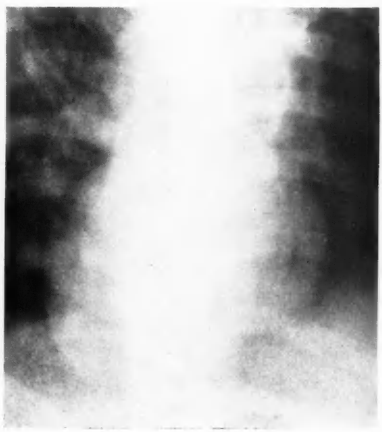


FIG. 44

completely obstructed by malignant growth; no previous difficulty in swallowing. Gastrostomy performed.

Male, aged 55. He had previously been under me for carcinoma of the œsophagus, and after radium treatment was swallowing well. After several weeks he came up much worse, unable to take anything but liquids, and I naturally supposed the growth had recurred. On examination I found at the site of the growth, 22 cm. from teeth, a moderately firm fibrous stricture, covered with normal epithelium, and in it a small wedge of decomposing meat (confirmed by section). The stricture was dilated, without producing bleeding, and he is is doing well.

Male, aged 36. Subject of an old lye stricture, at 34·5 cm. A mass of meat occupied 9 cm. of the œsophagus above this.

Dentures. Four Cases.

Male, aged 33. A denture carrying two incisor teeth lodged at level of top of sternum. Under direct œsophagoscopy it was found that a metal hook was firmly fixed on the left wall of the œsophagus, the pin from which a third tooth had been broken being fixed in the right wall. A "double internal version" manœuvre was needed to disimpact the two hooks, the wall of the gullet being lifted away, and afterwards protected, by means of the lip of the œsophagoscope. At the moment when this had been accomplished, and before the denture could be seized, the patient vomited, and swallowed it down; it was passed *per vias naturales* four days later. (Fig. 21.)

Male, aged 49. A large upper denture impacted in the œsophageal entrance, and removed under general anæsthesia.

Female, aged 26. Denture in thoracic entrance. On attempting to seize it, it slipped down and was passed later.

Male, aged 48. A large upper denture lying in the hypopharynx for seventeen days, the upper part visible by indirect laryngoscopy. Removed under local anæsthesia. (Fig. 23.)

Pins. Four Cases.

Age 9 months. Baby noticed to have difficulty in swallowing, and pin then missed. No dyspnœa. Radiogram shows pin lying open, to right of mid-line, point upwards at level of hyoid. Removed by direct pharyngoscopy. (Fig. 35.)

Age 9 months. Swallowed a safety-pin thirteen days ago. Brought up with extreme dyspnœa, and much swelling of neck, and Mr. Angell James performed tracheotomy; three days later he removed the pin through an incision in the neck swelling, only the head of it being then in the lumen of the hypopharynx. Recovery.

Age 3 weeks. See page 113. (Fig. 37.)

Age 10. A drawing-pin sticking in the mucosa of the larynx, removed. (Fig. 36.)

Toys. Four Cases.

Age 1. Swallowed a " tin horse " two days before, can take fluids only. A small tin goat found at 13 cm. and removed. (Fig. 38.)

Age 6. A brass disc the size of a farthing removed from the level of the sterno-clavicular joint.

Age 11 months. Swallowed his tin watch two days before, now will not take anything. Watch removed from œsophageal entrance; ulceration of mucosa on either side where it had rested. Recovery. (Figs. 39 and 42.)

Age 3. Swallowed the key of a clockwork mouse. It lay with the two wings level with the top of the sternum, and the shank upward. Removed from this position under general anæsthesia, 45 seconds. (Fig. 40.)

A CASE OF PRIMARY SARCOMA OF THE SPLEEN.

BY

ARTHUR L. TAYLOR, M.D.,
Pathologist, Bristol General Hospital.

MALIGNANT disease of the spleen is a rare condition. This very vascular organ might well be expected to show deposits of growth in cases where there is widespread dissemination of malignant cells by the bloodstream, yet such deposits are extremely seldom seen, a fact which is without doubt related to the destructive function exercised by the splenic pulp. Primary growths, though rather more common than secondary, are still to be regarded as pathological rarities. For this reason the following case of primary sarcoma recently encountered at the Bristol General Hospital is considered worthy of record.

Case Report.—On 19th January the patient, a woman of 54 years, attended the Casualty Department of the Hospital complaining of severe epigastric pain which had started quite suddenly three days before and had lasted continuously ever since. For the last month she had suffered from general weakness and some shortness of breath on exertion, but there had been no previous acute attacks. The pain was griping in character, but did not cause vomiting; there had been no hæmatemesis or melæna. Preliminary examination revealed an enormously enlarged spleen, and the patient was admitted forthwith for full investigation.

On admission the patient was a thin, pale woman with a

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On admission the patient was a thin, pale woman with a

slightly icteric tinge of the skin and conjunctiva. Temperature 99°, pulse 89, respirations 20, blood-pressure 120. Careful examination failed to reveal any gross organic disease of the heart, lungs, alimentary system or of the central nervous system. In the abdomen a large swelling, obviously the spleen, was found extending well across the middle line and almost as low as the pubes. The anterior notch could be well made out, and the surface, easily palpated through the wasted abdominal wall, felt nodular to the touch. There was no increase in size of the liver, and no ascites or œdema: the lymph glands throughout the body showed no enlargement.

The case, then, was one of massive splenic enlargement associated with evident anæmia, and an interesting problem in differential diagnosis was presented.

A differential blood-count gave the following results: Red cells, 3,480,000 per cmm.; hæmoglobin, 40 per cent.; colour index, 0·58 per cent.; leucocytes, 7000 per cmm.; neutrophile polymorphs, 67·5 per cent.; eosinophile polymorphs, 2·0 per cent.; basophile polymorphs, 1·0 per cent.; lymphocytes. 25·5 per cent.; large hyalines, 4·0 per cent.

These findings indicate an anæmia of secondary type, and served at once to exclude polycythæmia, pernicious anæmia and leukæmia, as well as any of the acute infections in which a leucocytosis or leucopenia is to be found. Considering the possibility of lymphadenoma, the differential leucocyte count was of importance, since in this condition there is commonly a considerable relative increase in large hyaline cells. No such increase was found; moreover, the absence of lymph gland enlargement and the enormous size of the spleen were sufficient in this case to negative a diagnosis of Hodgkin's disease.

A further possibility to consider was the splenomegaly associated with acholuric jaundice. This condition, in which the spleen sometimes reaches enormous proportions, is characterized by two peculiar features; an increased fragility of the red cells and the appearance of colloidal bilirubin in the blood-stream. In the present case the red cell fragility was normal, and Van den Bergh's reaction, both direct and indirect, was negative.

Gaucher's disease, in which the splenic enlargement is due to extreme lipoid infiltration of the endothelial cells of the splenic pulp, was put out of court because in this condition a similar lipoid infiltration of the liver and lymph glands produces an associated enlargement of these organs.

There was, then, remaining that ill-defined type of splenomegaly referred to as "splenic anæmia," a late stage of which is represented by Banti's disease, where the long-continued portal irritation has resulted in cirrhosis of the liver. It was thought, however, that supposing the case to be one of splenic anæmia, the tremendous size of the spleen could only mean a very advanced stage of the condition in which hepatic involvement must already have occurred; yet evidence of cirrhosis was entirely lacking. Splenic anæmia was therefore thought to be an unlikely diagnosis.

None of the suggestions outlined above appeared to be in agreement with the clinical and pathological features of the case, and the possibility of new growth had therefore to be considered. There were two reasons for which this diagnosis was considered likely. One was the nodular character of the surface of the spleen revealed on palpation. The other was the quite remarkable diminution in the size of the organ following radium application. In all 110 milligrams of radium were applied over the tumour for a period of eight hours; within a few days the spleen was so reduced in size that its lower pole could be felt no more than a hand's breadth below the left costal margin.

Operation was accordingly thought advisable, and splenectomy was performed by Mr. Moore on 14th February. The spleen was adherent to the liver and slightly to the diaphragm, but was removed without difficulty. Hemorrhage was negligible.

At operation no evidence was found of cirrhosis or of ascites, and no secondary deposits of growth were discovered in the abdomen. The patient made a good recovery, and for a time, although the anæmia persisted, she appeared to be doing well. Later, however, she developed a pericardial and left pleural effusion, and chest signs which were thought on both clinical

and radiological grounds to indicate metastatic deposits in both lungs. Unfortunately, the patient has since died outside the Hospital, and no post-mortem examination could be obtained to confirm this observation. As to the neoplastic character of the spleen itself there could, however, be little doubt.

Morbid Anatomy and Histology. The spleen on removal was a considerably enlarged organ of firm consistency, weighing 34 ounces, although when the patient was first seen it must have been at least three times this size. Numerous small nodules can be seen protruding beneath the capsule (Fig. 1), and the anterior margin shows a large pale infarct evidently of quite recent formation. On section the cut surface is found studded with these nodules, which are of the same homogeneous pink colour as the surrounding splenic tissue and barely distinguishable from it except on close inspection. (Fig. 2.) The Malpighian bodies are of insignificant size and widely separated from each other.

The pink, fleshy appearance of the gross specimen is strongly suggestive of neoplastic tissue, and this diagnosis is confirmed on microscopic examination of sections removed from various parts of the organ. Histologically it is at once evident that the great size of the spleen is due entirely to a massive overgrowth of the elements of the splenic pulp; the Malpighian bodies are few in number and atrophic, and play no part in the neoplastic process. In the pulp the endothelial structures are greatly increased; the blood channels are large but poorly formed and lined by cells much larger and much more deeply staining than those normally present. (Fig. 3.) In places the endothelial lining is deficient, in others it is several cells thick. Scattered in large numbers through the irregular reticulum are large hyperchromatic cells, some of them of giant size, containing several closely-packed nuclei or one large vesicular nucleus staining intensely with the basic dye. (Fig. 4.) The microscopic appearance of the discrete nodules seen in the gross specimen differs little from that of the intervening pulp, except that in them there is even greater cellular

PLATE XIII.

FIGS. 1 AND 2.

Surface and cut section of the spleen after removal. Multiple nodules are seen scattered beneath the capsule and through the splenic substance. The anterior border shows a large infarct caused by occlusion of a branch of the splenic artery, probably by the growth of one of these nodules in one part of its course.

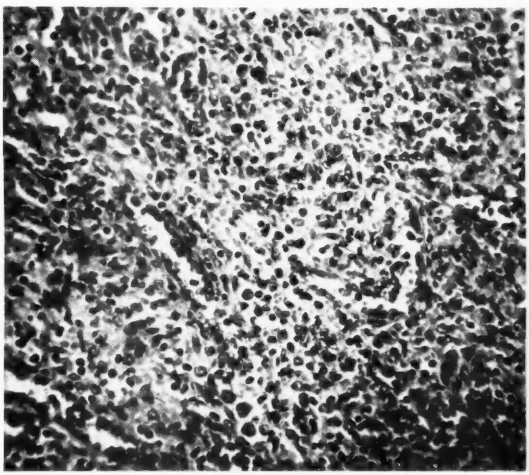

FIG. 3.

Microscopic section of a portion of the splenic pulp, showing ill-formed vascular channels lined by hyperchromatic endothelial cells of large size. Similar cells are scattered irregularly through the section, completely destroying the normal architecture.

PLATE XIV.

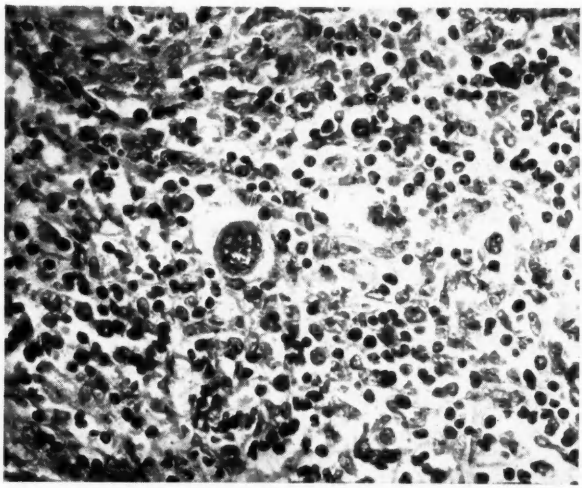

FIG. 4.

Higher power microphotograph of a portion of the splenic pulp, showing a large giant cell of obviously malignant aspect, containing a single deeply-stained vesicular nucleus. The neoplastic character of the surrounding tissue is very apparent. The lumen of the thin-walled vessel seen in the upper left-hand corner is partly filled with actively proliferating cells.

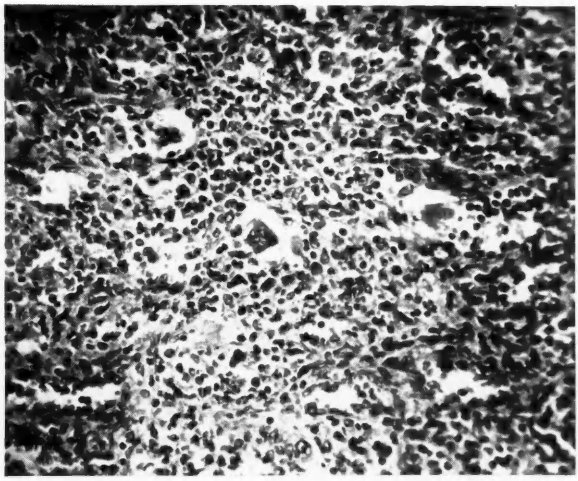

FIG. 5.

Microscopic section of one of the nodules described in the text, showing a similar general structure to that of the intervening tissue, but containing many more of the irregular multinucleated giant cells.

proliferation and the giant cells are much more numerous. (Fig. 5.) Many mitotic figures are present in all parts. The whole picture is one of sarcomatous growth arising in the endothelial cells of the splenic reticulum and producing a massive diffuse enlargement of the organ, in which the discrete nodules represent foci of particularly active growth.

Classification of the primary malignant growths of the spleen is a matter of some difficulty. Since Weichselbaum (1881) first described the condition few cases have been recorded, and Aschoff (1921) in his comprehensive text-book of pathological anatomy dismisses the subject in very few words. Ewing (1928) has collected a number of cases from the literature, and classifies them in the following three groups :—

1. Spindle-cell sarcoma: occurring as a comparatively benign circumscribed tumour, and microscopically showing spindle and round cells with trabeculæ and solid areas of fibrous tissue.

2. Endothelial sarcoma: producing as a rule multiple nodules in a greatly enlarged organ. This tumour is quite malignant, visceral metastases being commonly found. The structure consists of large irregular cells with single or multiple vesicular nuclei often with giant cell formation.

3. Primary lymphosarcoma: arising in the lymphoid cells of the splenic follicles and producing diffuse enlargement of the organ, sometimes with regional metastases. Histologically the neoplastic cells are of the type of large or small lymphocytes.

The largest of these groups is the second one, to which the case here recorded undoubtedly belongs. In this group the new growth is regarded as taking origin from the endothelium of the splenic pulp (Bunting, 1923), or from the endothelial cells of the splenic follicles (Foix and Roemmele, 1912). In the

present case the excessive overgrowth of the sinus endothelium and the atrophic character of the follicles indicate clearly an origin in the splenic pulp. On the other hand, in Foix and Roemmele's case, as well as in a similar case demonstrated by Professor Hadfield at a recent meeting of the Pathological Society, the Malpighian bodies were greatly enlarged and stood out with remarkable prominence on the cut surface of the organ. This group, then, would appear to contain at least two types of growth, although the endothelial origin is clear in each case.

Unfortunately, in the present case the occurrence of metastatic deposits lacks confirmation. It seems likely, however, that secondary growth took place, considering the well-recognized tendency of these tumours to metastasise and the rapid clinical course pursued in the present instance; the patient was dead within three months of the commencement of her illness.

My best thanks are due to Dr. J. A. Birrell, Dr. Carey Coombs and Mr. Clifford Moore for kind permission to make use of their notes.

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CLINICAL DIAGNOSIS OF PRIMARY SYPHILIS.

BY

A. MICHAEL CRITCHLEY, M.D.

THE tendency of the modern practitioner is to rely more and more on pathological and X-ray findings, to the exclusion of clinical investigation in arriving at a diagnosis. Whilst the undoubted value of the pathologist and radiographer, as aids to diagnosis, is not disputed, it must be remembered that they are only "aids" and are not indispensable, and that clinical observations are still the key to correct diagnosis. The present-day syphilologist often tends to subordinate his own clinical findings to the frequently confusing reports of the pathologist, much to the detriment of his patients.

In order to get the best results in treating syphilis the infection must be diagnosed before the Wassermann reaction becomes positive, and treatment must be started without delay. When a patient presents himself suffering from a venereal sore the problem is to find out as quickly as possible whether or not this sore is syphilitic, and to apply immediate treatment. According to Stokes,[1] the diagnosis of syphilis is a laboratory problem, and clinical appearances are worthless. Thus Stokes when confronted with a sore employs the following routine. A dark-field examination of the sore and adjacent glands is made, and local and blood complement fixation tests are performed. If the dark-ground examinations fail to

reveal spirochætes after repeated attempts, Stokes does a Wassermann follow-up; that is, the blood is tested weekly for four weeks and monthly for four months, at the end of which period, if the blood is still negative, it is then presumed that the sore was non-syphilitic. But what a tragedy if the final Wassermann is positive — four months wasted, during which the infection has become disseminated throughout the body. This generalization could have been prevented if a clinician who had the courage of his convictions had seen the patient in the first instance. Stokes claims that 82 per cent. of chancres have demonstrable spirochætes, which is a most optimistic finding; 42 per cent. would be a much more correct estimate. Again and again one sees a report on a scraping "No spirochætes found," only to be greeted with a second report a few weeks later, "Blood Wassermann++." It must be emphasized that once the Wassermann reaction has become positive it is impossible to guarantee cure, irrespective of the amount of treatment given. When a syphilitic sore is treated before generalization has set in, a cure, provided the treatment is adequate, can safely be guaranteed.

The clinical features of a genital chancre vary slightly according to the exact situation of the sore, but wherever situated there are always certain findings common to all. The most important item in examination is inspection. As soon as one starts feeling the sore, mistakes become more liable. Induration, which is such a characteristic trait of primary sores, can be seen more surely than felt, for all the normal folds of the skin around the sore become obliterated owing to the swelling and induration. The sore is usually painless. When the chancre is

secondarily infected, it can be seen by the way the patient manipulates his penis that it causes but little discomfort. Erosion is more common than ulceration. An ulcerative chancre has regular non-undermined edges, it does not appear to be healing in one pole and spreading in the other like an *ulcus molle*, and the base is covered by a serous non-purulent exudate. Bilateral inguinal adenitis is usual. It is a mistake to say that chancres are rarely multiple, for in about 25 per cent. of cases there is more than one primary sore. Many chancres are missed because gonorrhœa conceals the syphilitic lesion ; especially is this mistake made in meatal sores, and in patients being treated for gonorrhœa who subsequently develop syphilis which they acquired at the same time as the urethritis. The genitalia of each venereal patient should be examined every time he attends for treatment.

The easiest sores to diagnose are erosive chancres occurring on the inner surface of the prepuce and on the corona most commonly near the frænum. In these cases, owing to the induration the foreskin rolls back in an absolutely pathognomonic manner in one big piece, and the normal folds of mucous membrane are entirely obliterated.

Sometimes a chancre becomes secondarily infected, and phagedænic ulceration may result. In this type of sore a search for spirochætes is waste of time, and until the Wassermann becomes positive the diagnosis must be made solely on clinical observation. Severe pain is absent, and there is comparatively little inflammatory reaction of the skin. If the phagedæna is due to a soft sore, which is a rare occurrence compared with syphilitic phagedæna, the skin reaction is marked. A bubo in the groin in these secondarily infected ulcers is no contra-indication to a diagnosis of syphilis, for

there is no reason why some of the secondary organisms should not travel along the lymphatics to the inguinal glands and there provoke suppuration.

Intra-urethral chancres are another source of difficulty, but fortunately it is extremely uncommon for a sore to be entirely intra-urethral, as usually one pole of the sore is external to the meatus. Again, the absence of the normal folds of mucous membrane is to be observed. Digital examination if done carelessly will miss any induration, for as a rule it can only be felt in the long axis of the urethra and not from side to side.

Sometimes a thorough examination of a sore cannot be made owing to the presence of phimosis. These cases can still be diagnosed clinically without having to resort to surgical measures such as dorsal slit. If the sore is a chancre there is a definite bulging of the prepuce on that side, but there is neither reddening nor tenderness of the foreskin. On palpation induration may be felt.

Differential diagnosis of chancre includes the consideration of such lesions as *ulcus molle*, *herpes preputialis* and scabies. Provided each sore is examined carefully there should be a little difficulty in arriving at the correct diagnosis. Pathological examinations in these cases are useful if positive, but if negative they simply confuse and introduce an element of doubt.

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PORTAL PYÆMIA FOLLOWING DIVERTICULITIS, WITH REPORT OF A CASE.

BY

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THE rarity of portal pyæmia as a termination to diverticulitis makes the following case worthy of record :—

The patient, a stockbroker, aged 56, when first seen was a rather corpulent man of medium height, with a fresh complexion. He had always considered himself an exceptionally healthy man, and referred with pride to the great age attained by his parents. He admitted that when a young man there was a period when he drank whisky heavily, and up to the date of his present illness he had been accustomed to consume a moderate amount of alcohol.

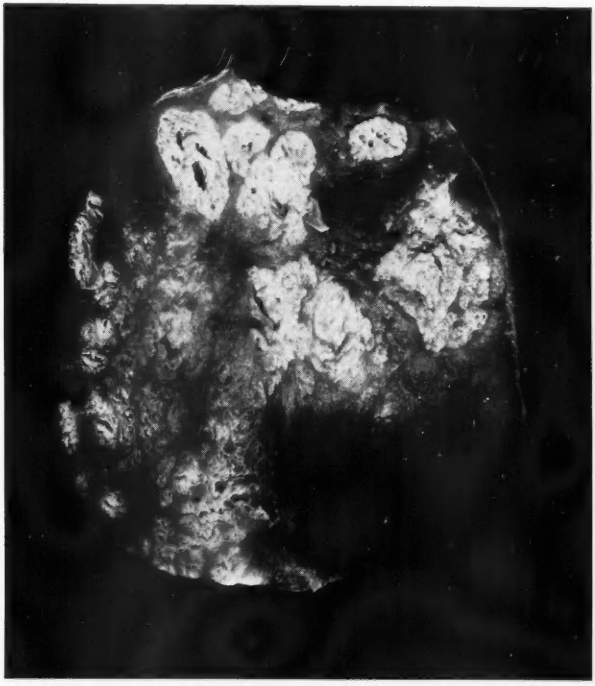
He first consulted one of us in 1925 for a short cough ; he had some pharyngitis and acknowledged that he was a heavy cigarette smoker. Early in 1926 he complained of frontal headache ; on examination pus was seen in the right middle fossa. Under treatment the headache and discharge quickly cleared up.

History of present illness.—At the end of November, 1928, the patient consulted one of us on account of loss of appetite, a furred tongue that did not clear up with aperients, a dry cough, and profuse sweats. He had lost interest in his work and was listless and slept badly. On examination some râles

were heard at the right base and there was a slight rise in temperature. He was advised to stay in bed, but at the end of a week there was no improvement, his systolic blood-pressure had fallen, but he refused to stay longer in bed. On December 10th he complained of slight pain in the right hypochondrium localized over the site of the gall-bladder. His temperature was 102°. He was ordered back to bed and given felamine. Three days later his temperature had dropped to normal, and he got up on his own initiative. On December 22nd he was expectorating a considerable quantity of nummular yellow sputum ; he was obviously losing weight, he was still sweating at night, there was persistent evening pyrexia and some signs of consolidation at both bases. No tubercle bacilli were found in the sputum. During the first fortnight of January, 1929, though confined to bed, he continued to lose ground ; his face began to look pinched, the night sweats were more profuse, and the temperature swung to over 100° every evening. He still complained of vague pains in the abdomen. On January 13th Dr. Carey Coombs was consulted ; he found consolidation at the right base, a further fall in the systolic blood-pressure, and tenderness over the upper quadrant of the abdomen, not sharply defined but greatest opposite the tenth costal cartilage. The patient was advised to go into hospital for further investigation, and was admitted to the Bruce Wills Memorial Hospital on the following day. The sputum was examined by Dr. Fraser, who reported "Muco-purulent sputum, with many pneumococci and a fair number of m. catarrhalis. No tubercle bacilli found." The fæces were also examined, but no abnormal organisms were discovered. The chest was X-rayed by Dr. Bergin, who reported : "The right chest wall fallen in considerably ; masses of glands in right hilum. Left lung, blotchy opacities scattered throughout the lung like unresolved pneumonia. Both domes of the diaphragm move well, and there is no evidence of fluid at either base."

Ten days after admission the signs in the right lung were decreasing, the temperature was falling, and the appetite improving. Three weeks after admission the temperature was

PLATE XV.



Section through the right lobe of the liver, showing the widespread distribution of the pyæmic abscesses described in the text. The greater part of this lobe is completely destroyed, yet no abscesses were found on the left side. (Half natural size.)

normal, the blood-pressure had risen, and a cholecystogram taken by Dr. Bergin showed "a large gall-bladder emptying normally; gall-bladder filled in twelve hours; much larger than normal; no evidence of stones; rate of emptying is rapid." On February 11th the patient's temperature was normal, the pulmonary signs had completely disappeared, and the abdomen was free from pain. He was discharged.

Seen a fortnight later, he complained of some fatigue and dyspnœa after exertion, but he had no pain and no temperature, and he decided to return to his office. His wife subsequently reported that he was working half days at his office, but was very tired on returning home.

On March 18th he reported again, complaining of constipation, a furred tongue, short cough and loss of appetite, occasional sweats; temperature 100°. There was some tenderness over the gall-bladder area. He persisted in his work and was no better a week later; he had more pain in the abdomen, had vomited some watery fluid and was sweating more profusely; his temperature was 100°. The next day he vomited a large quantity of fluid, containing evidences of twenty-four hours' delay. His wife thought he had looked rather yellow, but no icterus was observed on examination. On March 28th he vomited a large quantity of bile-stained material, and complained of pain in the right hypochondrium. No signs were made out in the chest; there was tenderness over a mass in the neighbourhood of the gall-bladder and the temperature was over 100°. Dr. Carey Coombs was again consulted and confirmed the findings; the possibility of a cholecystitis was considered and Mr. Clifford Moore was called in. Mr. Moore did not regard the case as typical of cholecystitis, and recommended observation. The patient was admitted to the General Hospital.

After admission, the condition of the patient deteriorated, but the vomiting ceased. The induration over the gall-bladder persisted, and the patient had five rigors within a few days. Mr. Moore performed a laparotomy on April 8th, and found a perfectly normal gall-bladder; the right lobe of the liver was enlarged, although the appearance suggested neither cirrhosis

nor growth. Pus was suspected, but though the liver was explored with a large needle in many places, none was discovered. There were no adhesions suggesting a subphrenic abscess. After the operation a blood count demonstrated a secondary anæmia with considerable polymorph leucocytosis. Blood culture was sterile, and no malarial parasites were found in the blood. A skiagram revealed nothing abnormal. Rectal examination was negative.

The patient's condition grew worse, the rigors increasing in severity and occurring almost daily until his death on May 8th. A post-mortem examination was made the following day.

Post-mortem Examination.—The body was that of a wasted elderly subject. The abdomen showed a clean, almost healed laparotomy wound, behind which the omentum was found adherent to the anterior abdominal wall. The adhesions, which were of quite recent date, were separated without difficulty, displaying a thin-walled healthy gall-bladder and viscera which appeared at first sight quite normal. On lifting up the anterior border of the liver, however, the under surface of the right lobe was found to bulge slightly near the hilum, and on inserting a finger into the softened liver tissue at this point about half an ounce of thick yellow pus was evacuated. The liver on removal was seen to be somewhat enlarged, and section through the right lobe opened up numerous abscesses of irregular size and shape. These were strictly confined to the right side, the left lobe showing nothing more than fatty changes of the parenchyma. There was no trace of cirrhosis. The portal vein appeared quite normal on section, but some of its intra-hepatic branches contained septic thrombi continuous with the abscesses described.

The case was clearly, therefore, one of portal pyæmia, and a primary focus of infection was sought in the intestinal tract. The appendix was first examined as being the most likely source ; it was kinked and bound down by old adhesions, but showed no trace of active infection. The intestines were laid open and submitted to careful examination. The small bowel was normal in all respects ; externally the only abnormality

found in the large intestine was a slight thickening involving no more than three inches of the sigmoid colon. On opening the sigmoid the mucous membrane was seen to be congested and slightly œdematous, and numerous diverticula were found extending deeply through the muscular coats of the bowel. Some of these contained large concretions of inspissated fæcal material; from others dirty yellow pus exuded, and could readily be expressed in large amount. Section through the wall of the gut showed a chronic inflammatory thickening of the subserous coat round the bases of the diverticula, but there was almost complete freedom from adhesions to the surrounding structures.

The other abdominal organs showed little of note beyond the toxic changes which were to be expected; the spleen was enlarged, pale and very soft, and the liver and kidneys the seat of advanced fatty degeneration. In the thorax the condition of the lungs calls for comment. Both lungs, which were free from chronic indurative changes, contained numerous small abscesses, none of them more than half an inch in diameter and all of them situated immediately beneath the visceral pleura. These were of recent development and obviously due to septic infarction from emboli reaching the lungs by way of the hepatic vein. The heart was pale and flabby, but showed no sign of gross organic disease.

Culture and microscopic examination of the tissues removed at autopsy served to confirm the diagnosis. Cultures of the pus expressed from the diverticula contained *B. coli* with fæcal streptococci, and *B. coli* was obtained in profuse pure culture from the liver abscesses and from those in the lungs.

Microscopic sections of the affected part of the sigmoid show the typical lesion of a diverticulitis of long standing. The diverticula are distended with pus and cellular and fæcal debris, and in many of them the mucous membrane is largely destroyed. There is a widespread infiltration of all the coats with chronic inflammatory cells, and here and there are found scattered foci of polymorph leucocytes. The subserous coat is much thickened and fibrosed and its vessels greatly congested.

In the liver and lungs the pyæmic abscesses show a quite

characteristic histological picture. Many of those in the liver are of considerable standing, containing necrotic material in which the cell elements can no longer be distinguished. The pulmonary abscesses are of very recent date, probably only a few hours or at most a day or two old.

The findings thus make it abundantly clear that the primary lesion in the present case lay in a diverticulitis of the sigmoid colon; that over a considerable period of time septic emboli passing to the liver by the portal vein have produced multiple pyæmic abscesses in this organ; and that the septic process has eventually involved the venous return from the liver, producing terminal metastatic abscesses in the lungs. A remarkable feature of the case is the circumscribed character of the primary focus compared with the wide extent of the secondary lesions found post-mortem.

Telling,[1] in a paper read to the Royal Society of Medicine, has stated that metastatic abscesses are unusual sequelæ of diverticulitis. An extensive search through the literature of the last twenty-five years failed to reveal more than two cases.

The first was reported by Whyte[2] in 1906:—

A horse dealer, aged 65, a stout, well-built man, gave a history of only three weeks' illness. There had been excessive consumption of alcohol in the past. He had no symptoms beyond a feeling of extreme weakness. There was profuse perspiration, with a dry and coated tongue. There was no pain, tenderness or enlargement of the liver when he was admitted to hospital or at any time thereafter. During the nine days the patient survived in hospital eighteen rigors occurred. Slight jaundice was noticed the day before death.

Post-mortem examination disclosed a left lobe of the liver full of abscesses, varying in size from a marble down to a pea, and containing pus swarming with *B. coli*. A segment of the

sigmoid colon eight inches long was found in a state of diverticulitis; one perforation into the peritoneum had been sealed off.

This case resembles the subject of our report: a man of similar build, constitution and apparent indulgences. The history, however, is shorter, and there were even fewer localizing symptoms. The appearance of abscesses in the left lobe of the liver is rather remarkable.

The other case was reported by W. J. Mayo[3]:—

The patient had already had a colostomy performed for diverticulitis; he reported some years later with a single large abscess in the liver associated with cholecystitis.

There is some doubt in this case as to which was the primary focus for the liver abscess.

Interesting points in the problems of causation, diagnosis and treatment of these cases arise. The pathological process involved differs in no respect from that which may occur at any focus of suppuration; it is one either of septic embolism, as in the present case, or of ascending pylephlebitis, in which event the portal vein when opened is found to contain masses of suppurating blood-clot continuous with the liver abscesses. Why should metastatic abscesses be encountered so infrequently in diverticulitis? An important consideration in this regard is the widespread fibrosis which is constantly present around the primary focus of infection. This is often sufficiently extensive to produce a large indurated mass, which may indeed be mistaken for malignant growth; apparently the fibrosis in most cases effectually seals off the primary site and prevents extension of the mischief. It is perhaps significant that in the subject of our report this reactive fibrosis was relatively scanty. Further,

the rarity of metastatic abscesses may be due in part to the comparatively low virulence of *B. coli*, which is the infecting organism most commonly found. The suggestion appears reasonable that in these cases some other factor is at work, whereby the virulence of this organism is increased. We have already noted an alcoholic history in our own case as well as in those elsewhere described. May it not be that the factor of alcohol has played a part by lowering the general resistance of the patient, or more particularly by damaging the detoxicating functions of the liver and thus rendering this organ incapable of destroying the bacteria, once they have arrived there ?

The difficulty of diagnosis of portal pyæmia is well exemplified in the case under discussion. The diagnosis made was that of an atypical cholecystitis ; the same mistake was made by Lane and Austin[4] in two cases of staphylococcal liver abscess. Manson - Bahr[5] has pointed out the insidious onset of the disease, so marked in this case. He also speaks of sudden short attacks resembling malaria ; at one stage of our patient's career this disease was suspected. He calls attention to the chronically furred tongue, the night sweats, the anorexia and emaciation, and states further that pain is rare in the more chronic forms. Pain was never a prominent feature in our own case, and there has recently been at the General Hospital another instance of portal pyæmia where pain was entirely absent throughout the illness. In this case the patient had complained for two months of a cold sensation in the back and had had repeated rigors, but there was no actual pain, and the patient never felt ill except during the rigors. This man died very shortly after admission, and at autopsy the right lobe of the liver was found to contain a solitary large abscess two

inches in diameter; there was a diverticulitis and peridiverticulitis involving four inches of the descending colon. Manson-Bahr stresses the diagnostic significance of shoulder pain, but neither the present case nor the one just referred to exhibited this feature.

How far may laboratory findings assist in the diagnosis of portal pyæmia? The blood-culture in our case was negative, nor was any other result to be expected, seeing that the infective process was limited to the portal circulation. Hurst [5] speaks of a retrograde thrombosis of the splenic vein occurring in pylephlebitis. In such cases the infecting organism might reach the spleen and be cultivated from the fluid obtained by splenic puncture. But it seems unlikely that a positive result would be obtained except in the most advanced cases; moreover, as the present one shows, portal pyæmia may occur quite independently of a pylephlebitis.

One important indication is to be found in the differential blood-count. This examination in our case showed a secondary anæmia associated with a marked polymorphic leucocytosis, which increased steadily up to approximately 30,000 cells per cmm. just before death. A leucocytosis was also present in the other recent case referred to. This finding is of value in indicating gross suppuration, and in view of the vague abdominal signs and absence of any definite tumour in the regions usually involved, might suggest the liver as the site of abscess formation.

As to treatment, Holden and Moran [6] have published a case of pylephlebitis following appendicitis which was cured with intravenous mercurochrome. In our case, considering the massive involvement of the liver and lungs and the widespread destruction of tissue, it is in the highest degree unlikely that mercurochrome

injections would have been of any value by the time that the symptoms indicated the possibility of portal pyæmia. At this late stage, when the liver is riddled with abscesses, surgical treatment is equally valueless and the issue inevitably fatal.

Our best thanks are due to Mr. Clifford Moore and Dr. Carey Coombs for their assistance in publishing these notes.

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AN EPIDEMIC OF VIRULENT DIPHTHERIA.

BY

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AND

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THE comparatively small but seriously lethal epidemic of virulent or "malignant" diphtheria described in this article exhibited certain unusual clinical and bacteriological characteristics which were thought to be of sufficient general interest to warrant recording. The cases comprised a series of 40 in number from the middle of January, 1929, to May 14th, 1929. There were 22 males and 18 females. The ages ranged from twenty-five to two years, but 52 per cent. were under ten years, and, curiously, 25 per cent. were in their eighth year. The cases arose in a large and scattered area, and no particular school or carrier or milk supply could be proved responsible for the outbreak.

Symptoms. — The membrane was unusually extensive, involving tonsils, fauces, uvula and soft palate, and in some cases the naso-pharynx. It always presented a peculiar, slimy and semi-transparent appearance, very much resembling the scales of a fish. Microscopically, swabs were invariably reported negative for the Klebs-Lœffler bacillus, but were swarming with streptococci. An even more curious fact, which, however, is explained later by the result of post-mortem examinations conducted in two cases, was that direct microscopical and cultural examinations

of large pieces of detached membrane were also returned negative for the Klebs - Lœffler bacillus and positive for streptococci.

A constant feature was marked cervical adenitis and peri-adenitis, so that the neck presented a "bull-neck" appearance. There was always an extremely fœtid odour from the throat, and nasal discharge was always profuse and in most cases hemorrhagic.

Toxæmia was severe, as evidenced by the dullness and apathy of the patient and the appearance of definite purpuric spots on the skin and the occurrence of ecchymoses around the site of the serum injections.

Two fatal cases of laryngeal diphtheria occurred in our own private practices, and we have heard of another in that of a neighbouring practitioner. All these were fatal in one to three days.

Treatment.—On the assumption that these cases were regarded as a very virulent form of diphtheria dominated by a secondary streptococcal infection, the combined use of anti-diphtheritic and anti-streptococcal sera was instituted. As a routine 40,000 units of diphtheria antitoxin and 3,000 units of anti-scarlet-fever globulins (B. W. & Co.) were given intramuscularly on admittance. The diphtheria antitoxin was repeated in twelve hours, if there was any sign of extension of the membrane or increase in the toxæmia. The anti-scarlet-fever serum was repeated every twenty-four hours for three successive days. In some cases as much as 104,000 units of antitoxin was administered. The other items consisted in keeping the patient absolutely flat in bed and avoiding the slightest exertion.

Results.—Ten patients died and thirty recovered, the mortality, therefore, being 25 per cent. The

deaths were either early from toxæmia, or late from myocarditis as evidenced by dilatation of the heart, extra-systoles, " gallop-rhythm " and cardiac vomiting.

Palatal paralysis occurred in 17 cases (42 per cent.), ciliary paralysis in 5 cases (14 per cent.), and otitis in 7 cases (17 per cent.). Broncho-pneumonia occurred in one case, that of an infant of two years, and ended fatally. No actual paralysis of the lower extremities was seen, but two patients lost their knee-jerks. Oculomotor paralysis did not occur in any case of the series.

Post-mortem findings.—Professor Walker Hall, of the Department of Pathology in the University of Bristol, performed an autopsy on one of the fatal cases, that of a male aged eight years. He found superficial sloughs and hemorrhages in the tonsils, but the larynx and trachea were free from any typical croupous exudate. The lungs did not show any areas of consolidation, and the heart, including valves and myocardium, was normal. The alimentary tract, liver and kidneys did not present any feature of note.

Cultures from the upper surface of the tonsil yielded streptococci, but Klebs-Lœffler bacilli did not grow out. Cultures from the trachea yielded streptococci and atypical diphtheria bacilli. Blood cultures taken from the heart blood were negative in every instance.

Cultures from the upper surface of the tonsil were injected into one guinea-pig, and cultures from the trachea into another. Both animals died on the fourth day. Both showed hemorrhages into the pleuræ and hemorrhagic suprarenals. A third guinea-pig was injected with the material grown from the deeper part of the tonsil. This animal lived for

fourteen days, and did not show any evidence of diphtheritic intoxication.

Guinea-pig A was injected with an emulsion from the trachea and tonsils + 800 units of diphtheria antitoxin, and was killed on the seventh day. It showed an absence of any local lesion, but a few hemorrhages into the suprarenals.

Guinea-pig B was injected with an emulsion from the trachea and tonsils + 0·5 cc. of scarlatinal antiserum. This was killed on the seventh day, and showed entire absence of any local or general lesion.

Guinea-pig C was injected with an emulsion from the trachea and tonsils + 800 units of diphtheria antitoxin + 0·5 cc. of scarlatinal antiserum. This was killed on the seventh day, and showed no sign of local or general lesion.

These findings show that death was due to a mixed infection of streptococci and diphtheria bacilli. Diphtheria antitoxin failed to give complete protection, but this was obtained when anti-scarlatinal serum was added.

THE HISTOLOGY OF THE AORTIC WALL IN ACUTE RHEUMATISM.*

BY

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THE histology of the aortic wall in acute rheumatic infection has until recent years attracted very little attention, especially in this country. Recently much work has been devoted to the subject of acute rheumatism, and the aorta has received its due share, chiefly at the hands of American observers.

In the literature the changes in the aorta have usually been classified as an acute aortitis, and rheumatic fever has been ranged with those diseases giving rise to a similar condition, viz. the exanthemata, gonorrhœa, acute tuberculosis, etc. (Allbutt[1]). As far back as 1912 Klotz[2] drew attention to the occurrence of unusual changes in the media. He gives an account of three necropsies of consecutive cases of rheumatic fever in young patients. The intima was not altered, but the media was thickened by increase of vascular elements, œdema and perivascular infiltration, with abundance of leucocytes and plasma cells. He noted signs of fibrous repair, and mentioned the possibility of this change predisposing to aneurysm. Coombs[3] in 1908 notes the presence of whitish-yellow patches at

* This Essay was awarded the Martyn Memorial Pathological Scholarship for 1929.

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the root of the aorta in rheumatic carditis, which proved inflammatory in nature.

This paper is an attempt to describe the appearances observed in five consecutive cases of rheumatic fever. One case was studied in some detail and forms the basis of this description.*

Briefly, the cases are as follows:—

(A) F. G., boy, aged 13 years. Rheumatic fever six years before death; since then acute bouts. Died in acute exacerbation. *P.M.*—Chronic and acute mitral and aortic endocarditis, adherent pericardium, hypertrophy and dilatation of all chambers of the heart.

(B) W. L., man, aged 60 years. Brought to hospital *in extremis;* died next day—little history obtainable. *P.M.*—Malignant disease of root of lung. Old and recent valvular disease of aortic and mitral valves. Adherent pericardium. Chronic interstitial nephritis.

(C) Boy, aged 5 years. Died in acute exacerbation of carditis. Heart lesion of about six months' standing. *P.M.*—Recent endocarditis of mitral and aortic valves. Adherent pericardium.

(D) J. C., girl, aged 7 years. Heart lesion of two to three years' duration. Died suddenly with acute carditis. *P.M.*—Recent pericarditis, with acute and chronic mitral mural endocarditis.

(E) A. W., girl, aged 14½ years. History of rheumatic fever of three years' duration. Died suddenly with acute carditis. *P.M.*—Recent pericarditis and mediastinitis, with recent endocarditis of mitral and aortic valves.

It will be noted that all five cases showed evidences post-mortem of recent infection.

Macroscopically, dilatation of the aorta was noted in two cases. The intima showed in four cases raised

* All the figures at the end of the Paper were taken from this case, but they may be regarded as representative of the changes found in all the cases.

whitish-yellow patches, some of them linear in outline, others irregular with crenated margins, never exceeding half a square centimetre. These patches are found almost invariably in the first half-inch or so of the aorta, usually in the neighbourhood of the aortic valves. In one case a thickened patch was present in the neighbourhood of the innominate artery ; similar patches, however, were seen at the root of the aorta. These streaks and patches bear a naked-eye resemblance to the so-called "fatty streaks" observed frequently in cases of typhoid, tuberculosis, septicæmia, etc. This resemblance is, however, only superficial, and on closer examination it will be observed that the rheumatic patches are whiter, less glistening and fatty-looking, and have a harder consistence if an attempt is made to scratch them with the finger. This was well shown in a case, not included in this paper, where both types of patches were present. In rheumatic cases these patches tend to be irregular in outline rather than streaky.

Microscopically, in all the sections examined the intima was found thickened. The thickening, though present throughout, is not uniform in extent ; at one spot the intima gradually thickens and forms a well-marked "hillock," bulging into the lumen of the aorta, an appearance somewhat resembling the endarteritis deformans of the smaller arteries.

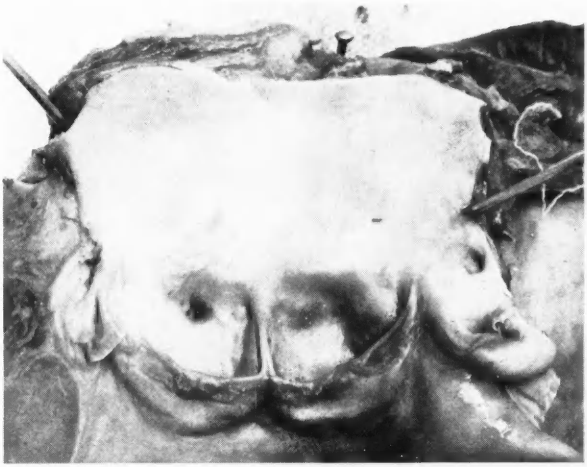
The endothelial lining in places is destroyed, probably as an artefact. In places it appears swollen and, most conspicuous of all, there are signs of endothelial proliferation in many sections, the endothelial cells being heaped up on the outer side of the endothelium. In many sections there is a

puckering, zigzag in outline, of the endothelial margin, usually most pronounced at the sides of the maximal intimal thickening. The intima, being probably œdematous before death, has shrunk in the process of preparation, the stretched endothelial lining now having to accommodate itself to the decreased superficies. On the whole, the endothelial picture is one of proliferation.

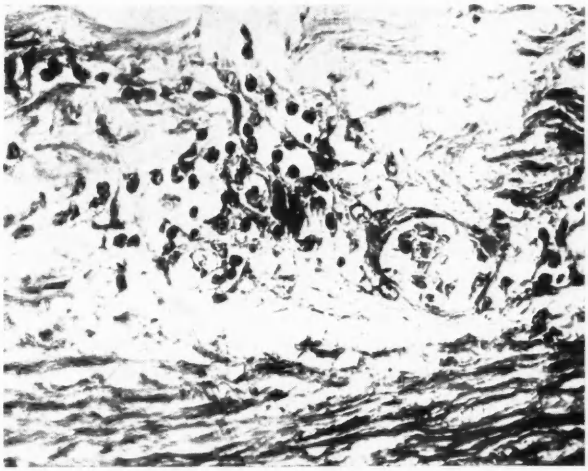
The intimal thickening is found to be composed of thick, wavy bands of collagenous material, running longitudinally as a rule, but in a few instances turning inwards or outwards. In places this degenerative matrix invades the media and is continuous with the intermuscular substance of the media, with the consequent destruction of the internal elastic layers. In this groundwork of the intima there are numerous open spaces, some large and irregular in outline, others small and rounded. As will be shown below, some of the smaller of these are fatty in nature, but the majority are not. Their true nature I have not found out. They may be due to fluid exudation. Attempts to demonstrate fibrin in them have not succeeded, but these have not been very exhaustive; moreover, a fluid exudate need not necessarily contain demonstrable fibrin.

There is considerable infiltration of the intima; the cells are mostly of endothelioid and lymphocytic type. The endothelial cells are in many cases present near the endothelium of the intima, from which they can be seen to be actively proliferating. The lymphoid are smaller, rounded, with a deeply-staining nucleus filling most of the cell-body, the latter staining faintly. The endothelioid cells are larger, of elongated shape

PLATE XVI.



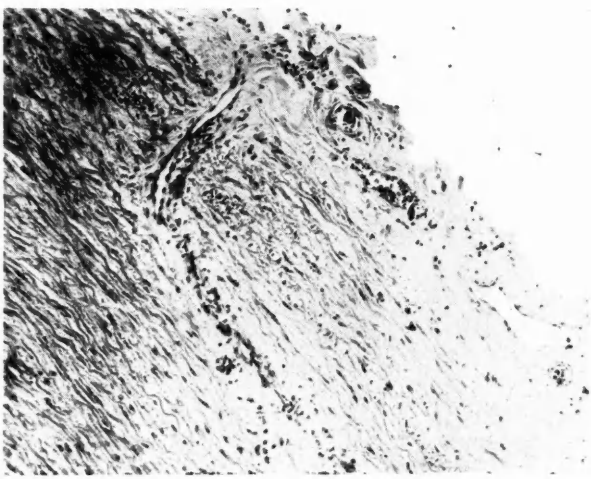
CASE A.—Aortic valves, with root of aorta. Shows some raised patches just above the valves.



CASE A.—Typical focal lesion in media. Shows vessel with lymphocytes and a few large endothelioids.

PLATE XVII.

Case A.—Orcein. Shows band of elastic to left of intimal "hillock," also absence at sites of medial lesions.



Case A.—Invasion of media by vessel and formation of node.

with blunt ends, and a pale nucleus with loosely-arranged chromatin.

I find the maximum cellular increase in the intimal "hillocks." Here they are mostly lymphocytes, but as we trace the intima round and it gradually decreases in thickness, the endothelioid cells come into the picture to the almost total exclusion of the lymphoid cells. All these cells are regularly distributed throughout the intima. In some cases the cells are placed in rows between the collagenous bands, forming the "palisade" appearance described by McCallum[4] in the auricular endocardium and by Pappenheimer and Von Glahn[5] in the aorta. This appearance has been the exception rather than the rule in the cases I have examined. This arrangement is due, no doubt, to the peculiar structure of the part, the collagenous bands when well formed pushing the cellular elements out of their course, so that they come to lie by their side. If the cells are too numerous this does not seem to occur. As has been mentioned above, when we trace the intimal "hillock" laterally we find that the spindle-shaped endothelial cells come to lie in rows parallel to the vessel wall. In sections stained with van Gieson there is well-formed fibrous tissue in this area. Some of the lymphocytes in all sections show signs of degeneration; their nuclei have lost their shape and become drawn and pyriform, while in some cases they are fragmented and lie free in the collagenous matrix. The endothelioid cells do not show such evident signs of degeneration; some of them possess two nuclei.

In some places the intima shows an appearance described by Pappenheimer and Von Glahn[5]; the

cells lie with their drawn-out pyknotic nuclei at right angles to the endothelial surface of the vessel. At one spot, in a section, there is a dense collection of cells, so that it stands out from the rest. In this agglomeration of cells lymphocytes mostly predominate; some in varying stages of degeneration. Some endothelials, round and elongated, are also present. In the centre of this cellular collection there are a few clear spaces. Just outside this lymphocytic zone, and on the outer aspects of the endothelium, there are a number of spindle-shaped cells, proliferating from the endothelial lining of the aorta, and making for this cellular zone; some are already around it. This appearance suggests an inflammatory response of a subacute nature. With toluidine blue sections the basophilic affinities of these cells are well demonstrated. Methyl-pyronin green-stained sections show an occasional plasma-cell in the intima.

Some sections were stained for organisms, but none were discovered.

Media and adventitia.—Many sections show a looseness and loss of the normal compact arrangement of the medial elements in its inner half. In places the collagenous matrix of the media is directly continuous with that of the intima through gaps in the internal elastic layers. There is an increase of the intercellular matrix in the inner half of the media; in some sections this appearance contrasts with the more compact outer half, whereas in other sections the whole width of the media presents this looseness in its architecture. In some cases the inner layers of the media are so altered that there is no abrupt transition into intima.

The muscle cells themselves vary in appearance; many of them appear healthy and take the stains well. Some, however, have lost their distinct cell outlines, nuclei have become less conspicuous, and in places there is a definite paucity of nuclei. The muscle cells are in cases thinned and atrophied as if compressed. In other sections the muscle nuclei, or in some cases fragments of them, lie scattered in the intercellular collagenous matrix. These changes are best marked under the greatest intimal thickening.

In the outer half of the media there is a different picture. The muscle cells and elastic fibres, in so far as they are present, appear normal; the compact arrangement is not grossly disturbed, and there is little, if any, increase of intermuscular substance.

Scattered throughout the outer third of the media there are areas of complete loss of the essential medial elements, muscle and elastic, and this is replaced by small focal lesions of an unusual character. These lesions present different appearances in outline and in histological structure. In some cases they are small and oval in outline, in others they consist of narrow bands lying circularly around the vessel wall. These appearances are no doubt determined by the histology of the part; as the branches of the vasa vasorum enter the media they invade it to a variable extent, usually to about its outer third only, and then divide into fine lateral branches which run circularly around the vessel; along these the lesions are apparently distributed. As they increase in size they are modified in their spread by the muscular and elastic fibres.

These foci differ in structure according to their

age and probably to the virulence of the infection. A typical focus consists of a small, thin-walled blood-vessel, usually containing some red cells. This vessel may show endothelial proliferation. Around it there is a collection of cells of two types; some are of the usual lymphoid type, with a deeply-staining nucleus filling the cell-body. The other cells are less regular in shape; they may be round, but are usually large and fusiform with blunt ends. They have a pale, anæmic-looking nucleus with loosely-arranged chromatin, which gives it a characteristic vacuolated appearance. In a few cases two or three nuclei may be present in one cell, arranged in a row along the long axis of the cell. Toluidine blue brings out the chromatic arrangement very well and the vacuolated appearance is very evident. These cells correspond in appearance and staining characteristics with those described by Coombs[6] as occurring in the submiliary nodules in the myocardium in rheumatic fever.

The whole lies in a structureless collagenous matrix which may contain remnants of muscle and elastic tissue. This ground substance is not sharply demarcated; at its periphery it insinuates itself between the muscle and elastic fibres, losing itself in the connective tissue substance of the media. In some cases the lymphoid cell predominates, in others only endothelioid cells are present, and these are seen proliferating from the vessel wall. In still other cases some spindle-shaped endothelioids are present, lying parallel to the muscle layers, and surrounded by collagen substance; an appearance suggestive of cicatrisation. Occasionally a small vessel is seen with a few round endothelial cells round it; there is no

collagen groundwork and no destruction of muscle or elastic tissue. I take this to be an early stage in the development of the node.

The adventitia is greatly thickened, so much so that it can be detected by the naked eye. It is mainly due to connective tissue and vascular increase. This vascular increase may be very prominent. It is mainly seen next to the media; the vessels are thin-walled, dilated and probably of recent formation. Some of the larger vessels show proliferative changes in their walls, of endothelial nature. This is so pronounced in some cases as to encroach into the lumen of the vessel, thereby giving it an appearance of endarteritis. The characteristic feature in the adventitia is the cellular infiltration; scattered throughout the adventitia are numerous cells, lymphocytes and endothelials, and occasionally plasma cells. This cellular increase is more marked round the vessels, some of these being completely surrounded by a "cuff" of cells.

In some sections we find evidence of the stages by which the medial lesions described above come into existence. As we trace a series of consecutive sections we find at one spot in the adventitia a highly-congested area. There are a few vessels with perivascular cell collections, chiefly consisting of spindle-shaped endothelials; opposite this in the outer layers of the media there is a gap in which the muscle and elastic elements have disappeared, and this is occupied by an extension of collagen substance from the adventitia. A few spindle-shaped endothelials may be arranged along this gutter. As we trace the sections on, we find that a small thin-walled blood-vessel occupies this space; by its

side and parallel to it are usually some spindle-shaped endothelial cells; farther on in the series of sections this vessel leads to a small focal lesion in the media. It is not uncommon to find more than one example of this nature in one section.

In other sections the adventitia is thickened by dense, wavy bands of connective tissue with very few cells. Near the media there is a proliferation of the vasa vasorum, some of the vessels showing endothelial endarteritis. Along the inner parts of the adventitia there are strands of muscle fibres and elastic tissue parallel to the medial layers and of a ragged and diffuse nature; they are probably the outer medial layers disturbed from their normal relations by the intense vascular invasion.

Orcein-stained sections show an increase in the elastic tissue elements of the intima. This consists of a fairly thick band of elastic fibres running along the intima and occupying its middle third, in the position of the so-called "stripe." As we trace this elastic band round, it almost totally disappears at the intimal "hillock," where degenerative changes appear greatest. In some sections no evidence of this band is present; in others there are traces of it in the form of fine, short fibrils running across, or rather skirting this area and taking a curved course around it. In places this band seems to be pushed into the intimal elastic lamina by the degenerative process here. It is an interesting fact that, on the other side of the intimal "hillock," no such band is present.

There are some fine elastic fibrils along the inner branch of the intima just external to the endothelium. The ground substance of the intimal "hillock" stains

a greyish-pink colour with fuchsin; lateral to this the thickened intima takes on a deep pink along the middle third. However, in many sections there is not much elastic tissue increase. The internal elastic lamina presents different appearances; in some sections it is intact, in others it is broken up, stretched or distorted. It may present merely a series of short elastic fibres, often fenestrated by the collagenous matrix of the intima as it invades the media.

In the inner third of the media we find the elastic fibres separated from each other by well-formed collagenous tissue; the elastic laminæ have in some cases lost their parallel and regular appearance so characteristic in the normal aorta. The elastic fibres may be torn and be present only in short shreds throughout the whole thickness.

In the outer medial layers there is a complete absence of elastic tissue in the areas occupied by the medial lesions; the laminæ are abruptly broken up at the margins of these areas, and only in a few cases do I find traces of elastic fibrils traversing them. In other instances the elastic tissue is pushed aside by these medial foci. Some elastic fibres are seen in the adventitia, next to the media; they are in short shreds, and probably belong to the media mentioned before.

In a few sections stained with Sudan III. and Scarlet Red small globules of fat can be seen scattered about the intima, mostly at its thickest portions. In the deep part of the intimal "hillock" and in the adjacent areas of the media there is a large deposit of fat, consisting of globules of varying sizes; these have fused together in the centre forming a large

mass. In places these fatty globules extend deep into the media, occupying spaces between the muscle fibres and pushing them aside, thereby accounting in some measure for the looseness of the muscular cells seen in the hæmatoxylin and eosin sections. Many of the muscle cells themselves contain fat droplets, these being present especially in those cells near the fatty deposit, and this is probably the initial step of the degenerative process; as the fat gradually fills the cell the latter disappears and the fat comes to lie in the intermuscular spaces. The collection of fat is fairly localized, and as we trace it laterally it gradually disappears, as the intimal "hillock" passes into the less thickened portions of the intima.

In other sections a striking appearance is seen. Large, irregular collections of fat are present in the sub-intimal layers of the media; there is little, if any, in the overlying intima. The internal elastic lamina may be unaltered, and course over the fat deposit undisturbed. That such localized collections of fat are present is not to be wondered at; Andrewes,[7] writing on arterial degeneration, says, "Whereas the slighter degrees of fatty changes are usually diffuse, the more severe degrees tend to be patchy in distribution. Such local foci of degeneration are situated remote from the vasa vasorum."

CASE B.—The intima shows the puckering noticed in the other sections. There is much fibrous tissue in the intima; the musculo-elastic layer is present in sections. The elastic tissue of the intima is also increased. The internal elastic lamina is not easy of recognition, making the transition from intima to media difficult to localize.

In the media there is considerable increase of fibrous tissue; the elastic is present as very fine shreds. The muscle fibres are greatly diminished in number. These facts are very striking in orcein sections. What muscle there is is grossly disturbed; the nuclei of the cells appear fragmented and drawn out with their long axes pointing in all directions, instead of the familiar parallel arrangement. In the outer layers of the media there is a focus of cells similar to that described above. The adventitia shows a few perivascular collections of cells.

CASE C.—The intima is slightly increased in thickness. The media in some sections shows increase of intermuscular connective tissue; the muscle cells show somewhat similar changes to the previous case, their nuclei lie scattered irregularly throughout.

The deeper layers of the media are interrupted by the focal lesions previously described. In this case the lesions are very vascular, some of them in a small area contain two or even three thin-walled capillaries; the cells are few. There is much collagenous material around them.

In the adventitia there is fibrous and vascular increase; some of the adventitial vessels exhibit endothelial proliferation showing a well-marked appearance of endarteritis. These vessels have usually a "cuff" of endothelial cells surrounding them. The perivascular cellular increase is very marked; in addition to the two common types of cells found hitherto, *i.e.* endothelioids and lymphocytes, there are here large cells, usually polygonal, occasionally elongated with a more or less central nucleus. The cell takes the eosin fairly deeply, and the nucleus,

though taking the hæmatoxylin well, has a loose chromatin structure which makes it appear vacuolated. These cells are larger than plasma cells, and resemble the giant cells seen in the rheumatic nodules in the myocardium etc., except that in no case could I see more than one nucleus.

CASE D.—Here the intima was not much altered. The media showed similar focal lesions as hitherto described. In this case these foci are rather cellular; a long band of collagenous matrix, extending circularly round the vessel for a considerable way, may only contain a blood vessel and a few endothelial cells round it. This collagenous substance is usually found extending from the adventitia. In orcein sections an increase of the intermuscular matrix is found similar to the previous cases.

The adventitia shows a rich vascularity, and some of the vessels show intimal endothelial proliferation. Round the vessels and scattered throughout the adventitia are numerous cells; endothelials, monocytes and many plasma cells. These cells were not seen in such numbers in any of the other cases. It is interesting to note that in this case plasma cells are a predominant feature in other organs.

CASE E.—There is some intimal increase. In the media there are again the focal lesions previously described. As in case D, these are not very cellular. The adventitia shows cellular infiltration round the vasa vasorum. Orcein sections show an increase of fibrous tissue in the intima and the media.

COMMENT.—Two apparently different appearances, that in the intima and that in the media, are to be accounted for.

What relation, if any, has the intimal change to the underlying pathological process in the media? Klotz[8] has said: "We believe with Koester that, in as far as the inflammatory processes of the media associated with rheumatism are concerned, the intimal thickening is secondary." Pappenheimer and Von Glahn,[5] on the other hand, regard the intimal changes as a diffuse reaction to infection from the lumen. It is scarcely possible that the character of the intimal increase as described is wholly of the nature of a compensatory process as Klotz suggests. The appearances indicate an inflammatory response of a subacute nature, to an infection from the blood-stream. Whether this response is specific for rheumatic fever it is scarcely possible to say. Pappenheimer and Von Glahn believe it to be so. They describe, what they regard as characteristic, the formation of "palisades" and the presence of Aschoff cells. I have not been fortunate in this last respect. I have seen the "palisade" appearance in the intima, but only in a few sections. Moreover, I have seen typical "palisades" in the intima of the aorta in a case of acute tuberculosis. The intimal change cannot, however, be wholly explained as inflammatory. I suggest that there is at the same time an attempt on the part of the intima to make good the damage done to the underlying media. These I believe to be strongly supported by the increase in fibrous and elastic tissue; especially I refer to the elastic band running through a portion of the intima as described above. I do not know of any condition where elastic increase takes place as a response to an infection. It might be argued that the elastic increase is of the

nature of the normal "stripe" seen in the developing arteries. If this were so, one would expect to find this elastic tissue present in all sections and throughout the whole of the intima, whereas, as mentioned before, it is only present at one side of the intimal "hillock." Furthermore, the normal stripe in a subject of thirteen years is never of such thickness and density. This elastic increase corresponds with the "local hyperplastic sclerosis" of Andrewes,[7] which he describes as occurring at points of special stress. The origin of this elastic tissue is a much debated point. Jores[9] believed it to be the product of the internal elastic layers. Evans,[10] in his Goulstonian Lectures, mentions the possibility of the endothelium of the intima being responsible for the elastic production. The appearances in my sections seem to support this view. I have described the arrangement of flattened endothelials lying along the intima, and it is amongst these cells that the elastic band is found. Furthermore, it is difficult to believe that an internal elastic lamina so damaged as that in Case A could possess such vitality as the production of this elastic would entail.

The changes found in the inner layers of the media can be explained by the impairment of the nutrition of these medial layers, due to the thickening of the intima. Hence, here also in the media we find the fatty change most pronounced, the highly-specialized muscle cells bear the deprivation of nourishment very badly. The perivascular lesions found in the outer third of the media correspond with the findings of Pappenheimer and Von Glahn[5] in the aorta and Kugel and Epstein[11] in the pulmonary artery. One may hesitate to define these lesions as typical Aschoff

bodies, but there can be no doubt that the type of reaction here present is essentially that found in rheumatic manifestations elsewhere. The response is similar, only differing in degree and modified by the histology of the part in which it occurs.

Cases C, D, and E have been included to show how the appearance may be modified in degree, and also by the age of the lesion, these losing their cellular characters and finally cicatrising. Probably in many cases they leave little evidence of their former presence.

In Case B the findings might have been accounted for by the age of the patient and the renal changes present, but for the fact of the well-marked inflammatory foci in the media. We can exclude syphilis both macroscopically and microscopically: periarteritis nodosa never occurs in the aorta, and can therefore be disregarded. Moreover, the microscopical appearance of these focal lesions in Case E is similar to that found in the other cases, and may, therefore, be placed with them. This case emphasizes the fact that a rheumatic infection which has been dormant for many years may, even at the age of sixty, flare up into activity. The pathological features in the adventitia are to be expected if one recalls that through this the infective agent, virus or toxin, has to pass to reach the outer medial layers. The response is also characteristic of the rheumatic infection, being more generalized and diffuse owing to the rich vascularity of this part. Recently Barnard[12] described a case of rheumatic periaortitis; the appearance of the adventitia in Cases C and D might well be the initial stage of such a condition.

SUMMARY.

1. Five cases of rheumatic fever have been examined histologically.

2. In all of them lesions of a distinctive character have been found in the aortic wall.

3. These consist of areas of subacute inflammation around a small vessel, of a nature similar to those found in the pericardium, myocardium and other tissues in cases of rheumatic fever.

I should like to thank Dr. A. L. Taylor for allowing me to make use of specimens and for his kind help, also Dr. Carey Coombs for much encouragement, without which this would never have been attempted.

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[2] Klotz, *Trans. of Assoc. of Amer. Phys.*, 1912, xxvii., 181.

[3] Coombs, *Quart. J. Med.*, 1908, ii., 27.

[4] MacCallum, *Bull. Johns Hopkins Hosp.*, 1924, xxxv., 329.

[5] Pappenheimer and Von Glahn, *Jour. Med. Res.*, 1924, xliv., 489; *Amer. Jour. of Path.*, 1926, ii., 235; *ibid.*, 1927, iii., 583.

[6] Coombs, *Jour. Path. and Bact.*, 1911, xv., 489.

[7] Andrewes, *Report on Arterial Degeneration*, 1912.

[8] Klotz, *Jour. of Path. and Bact.*, 1913, xviii., 259.

[9] Jores, *Ziegler's Beitr.*, 1898. 24458.

[10] Evans, *Goulstonian Lectures*, 1923.

[11] Kugel and Epstein, *Arch. of Path.*, 1928, vi.

[12] Barnard, *Jour. of Path. and Bact.*, 1929, xxxii., 95.

Reviews of Books

The Treatment of the Sick Poor of this Country (Rogers Prize Essay, 1926). By H. J. McCurrich, M.S., M.B., F.R.C.S. Pp. viii., 132. London: Oxford University Press. 1929. Price 5s.—The wide latitude allowed by the judges of the Joseph Rogers Memorial Prize has been taken full advantage of by Dr. McCurrich. An Essay was asked for, and a captious critic might ask what is the limit of size of an essay. The author has composed enough for a book and was awarded the Prize. Most of the material of this book is a history of the medical service to the poor, taken from many sources and as an historical survey is of interest. We do not find that Dr. McCurrich has evolved any original constructive policy for the service, which is admittedly not fully satisfactory. This, we take it, was the intention of the founder of the Prize, and we fear that those who desire the betterment of the Poor Law medical service will not benefit much by the book. Neither are we always in agreement with the views that Dr. McCurrich expresses on the efficiency of the present state of affairs, but these are his opinions and the solution of some of the difficulties is obscure. Two slight errors have been detected. The Society of Merchants of Bristol never, as a corporate body, equipped or sailed ships from that Port. They acted more as a Harbour Trust, to use a modern term, renting the harbour at a small sum in return for the dock dues and an undertaking to build quays. The other is, the name of Dr. Rogers' more distinguished brother was not Theodore as given in the bibliography, but Thorold.

Tweedy's Practical Obstetrics. Sixth Edition. Edited by B. Solomons, M.D., F.R.C.P.I. Pp. xxii., 759. Illustrated. London: Oxford University Press. 1929. Price 25s.—This maintains the high standard of the previous editions, and embodies all the modern methods of practice and treatment associated with the Rotunda Hospital. Changes of opinion in cause and treatment have been indicated in the text, and this is seen in the treatment of accidental hemorrhage. Ante-natal care is not dealt with at much length, but this is made up for in that pelvimetry is very fully considered and well illustrated. The modern theories of toxæmia are all set out, and the very successful treatment adopted at the Rotunda fully described. The text is very readable and the illustrations good and easily understood. It is an excellent text-book.

Nephritis. By T. I. BENNETT, M.D., F.R.C.P. Pp. viii., 94. 2 Plates. London: Oxford University Press. 1929. Price 6s.—This monograph embodies the substance of the Goulstonian lectures delivered by Dr. Izod Bennett. The problems of uræmia, œdema and blood-pressure are discussed in the light of recent research in connection with nephritis. The author concludes with a most valuable and practical chapter on the treatment of nephritis, and diet scales and tables for constructing low protein diets are added. The main purpose of the book is a demonstration of the fact that nephritis is a general dyscrasia, and not simply a disease of the kidney. The importance of the extra-mural factor in Bright's disease is as yet very imperfectly appreciated by the medical profession, and this very seriously confuses the problems of prevention, classification and treatment. By his lucid style and skilful marshalling of facts the author has succeeded in infusing fresh interest and new life and shape into the study of this group of renal diseases.

"Terminal" Disinfection. By Professor C. CHAGAS. Pp. 45. Edinburgh: W. Hodge & Co. 1928. Price 2s. 6d.—In this excellent little book the Director-General of Public Health for Brazil sets forth clearly the arguments against the employment of terminal disinfection after acute infectious diseases. It must be pointed out that he does not in any way minimise the value of "current" disinfection, *i.e.* disinfection especially of bedclothes and other fomites carried out while the patient is going through the course of his illness. Dr. Chagas's argument is that the life of most pathogenic organisms outside the human body is comparatively short, and that they are probably dead by the time terminal disinfection is carried out at the end of the illness. Certain large towns in America and England have discarded terminal disinfection many years ago, and are perfectly satisfied with the results. The only criticism we would make upon Dr. Chagas's exposition of this theory is that, like a good lawyer, he stresses his own points to their utmost value and pays but little regard to possible counter-arguments. If the matter was quite so well proven as he would lead us to believe, it would not still be the case that the majority of large cities in the world still carry out terminal disinfection after the more serious infectious diseases, adopting as they do the attitude that while there is any doubt it is better to err on the side of safety. An instance of such doubt may be found in the recent

discovery by a number of different workers of hæmolytic streptococci in books which have been used by scarlatina patients. Nevertheless, we believe that the main principles laid down in this book are correct, and will probably ultimately become the generally accepted practice, though possibly in a modified form.

Parkes' and Kenwood's Hygiene and Public Health. Edited by H. R. KENWOOD, M.B., D.P.H., and H. KERR, M.D., D.P.H. Eighth Edition. Pp. xii., 823. Illustrated. London: H. K. Lewis & Co., Ltd. 1929. Price 21s.—One does not so much expect " Parkes and Kenwood " to follow a recognized standard as look to it to set one up. But no book, however ably it may be written, which deals with a series of activities so flexible and so progressive as those connected with the public health can long survive simply on its reputation. It must, in successive editions, keep up to date, and that is precisely what the eighth edition has done, and done admirably. The revision, as one would expect, bears the stamp of authentic practical knowledge and experience of present-day developments. Not only students, but those engaged in the routine work of a health department, will find the book full of suggestive and helpful information on almost every aspect of their work. Amongst the newer matters dealt with are Maternity and Child Welfare, Industrial, Marine, Tropical and International Hygiene, and the School Medical Service. Adequate descriptions are given of such comparatively recent measures as the Meat Regulations of 1924, the Milk (Special Designations) Order, the Preservatives in Food Regulations, the Puerperal Fever Regulations, and so forth. Some reference might have been made to the possible cerebral complications of vaccination, and perhaps to the Adoption Act, but any discussion on the effects of the new Local Government Act had to be left to the next revision. In a word, this edition has not only maintained, it has enhanced the reputation of what is universally recognized as a standard text book.

Manson's Tropical Diseases. Edited by P. H. Manson-Bahr, M.D., D.T.M. & H., F.R.C.P. Ninth Edition, revised. Pp. xx., 921. 35 plates, 401 figures, 6 maps, 34 charts. London: Cassell & Co., Ltd. 1929. Price 31s. 6d.—Medicine and our knowledge of tropical diseases do not stand still. This classic book has already been through eight editions in less than thirty years, each called for by advances in the knowledge

of diseases of warm climates, their etiology, diagnosis, course and treatment. This ninth edition, published four years after its predecessor, has its origin from the same cause. In it are set forth many new facts, also changes in our ideas with regard to some diseases, due to recent work. The most notable example is Yellow Fever. Its presence in West Africa has caused much concern of late years, and it has been under investigation for some time past. Special attention has been given to the subject of treatment, and the reader will find that of the various diseases full, complete, and explicit. Naturally, that of Malaria, the most frequent of tropical diseases, receives special attention. That suitable to each of the various types of the disease is pointed out. Special points for special cases are indicated, and where necessary emphasized. The protozoological and laboratory sections in the appendix has been thoroughly revised and brought up to date and a new section inserted on blood transfusion " suitable for employment under tropical conditions." Although some thirty new text figures with five half-tone colour plates have been added, the total number of pages in the book remains practically the same, and it thus retains one of its original aims, is " of handy size." In our opinion this manual of diseases of warm climates is full of sound, reliable and adequate information, and ought to be in the kit of all those who have to deal with tropical diseases.

Editorial Note.

Hospital Rag ended.

THE undergraduates of the University have resolved, after several discussions in the Union, to stop their annual collection for the Lord Mayor's Fund known as the "Hospital Rag."

The finances of our voluntary hospitals can ill afford to lose the contributions which have in the past been raised by this University effort. But the decision of the University Union is well-considered and fully justified. The mere title "Hospital Rag" probably offended more than it attracted, and it was calculated to bring unmerited reproach on the undergraduate collectors.

The time and energy expended by the officers of the Union in arranging the "Rag" were greater than any responsible authority should have dreamt of asking. If the Lord Mayors of this city had had any inkling of the dislocation of work and athletics caused by these preparations they would never have consented to be responsible for such a disturbance of routine.

An increasingly poor response to the collectors' appeals showed that public opinion in Bristol is out of sympathy with attempts at mass-coercion, which may sometimes succeed in less sophisticated communities. Moreover, the President of the Union was brought to death's door by illness contracted as the direct result of overwork and exposure before and during the "Rag." Mr. Cook is heartily to be congratulated on his recovery and the Union on the decision it has taken.

It remains for the next Lord Mayor to discountenance any revival of the "Rag" in other guise or

under other name. There is no possible justification for imposing on the undergraduates' good nature except, perhaps, the obvious advantages that with their good nature is combined impeccable honesty, and that they did not even deduct commission from their collections.

Obituary.

HERBERT GODFREY EYCOTT,

Assistant Librarian in the Medical Library of the University of Bristol.

THE death of Mr. Herbert Godfrey Eycott has deprived the University of the services of an Assistant Librarian whose work in the Medical Library was highly appreciated by the members of the Medico-Chirurgical Society. Mr. Eycott was appointed to the Medical Library in October, 1924, after holding a post in the Library of the Royal College of Physicians, London. He came, therefore, equipped with a wide knowledge of medical literature which he willingly placed at the disposal of readers in our library. The part he played in the production of this journal might pass unrecognized if the editorial committee were not to acknowledge his valuable help in proof reading, sending out and collecting books for review, and more especially in verifying references in original articles. The writers of most of the articles in this journal owe to Mr. Eycott an accuracy in quotation and reference which may sometimes have surprised themselves no less than it impressed their readers.

Mr. Eycott, who died at the early age of forty years, was educated in Leeds, and shortly afterwards took a position in connection with the railways of the south of Spain. At the beginning of the War he joined the Yorkshire Regiment and was twice wounded, on the second occasion so severely as to lead to the amputation of his right leg.

He bore his disability patiently and gallantly, and his final illness gave further evidence of his wonderful fortitude in the face of grievous pain and suffering. He died on June 5th, deeply mourned by all who knew him, leaving a widow to whom we offer our deepest sympathy.

Meetings of Societies.

Bristol Medico-Chirurgical Society.

The seventh meeting of the session was held on Wednesday, 10th April, 1929, the President in the Chair.

Dr. C. Dix showed a case of ichthyosis.

Dr. H. H. Carleton read a paper on **"The Clinical Significance of Sleep Disorders."**

Mr. J. Paul Bush, C.M.G., C.B.E., Ch.M., M.R.C.S., was elected an Honorary Member of the Society.

The eighth meeting of the session was held on Wednesday, 8th May, 1929, the President in the Chair.

Mr. Duncan Wood showed a case of fracture-dislocation of both astragali.

Mr. A. W. Adams demonstrated a specimen of double ureter.

Dr. F. Mayes showed skiagrams from a case of Paget's disease.

Mr. S. V. Stock read a paper on some observations on anæsthesia, which will be reported in our next issue.

Dr. Taylor read notes on **"A Case of Primary Malignant Disease of the Spleen,"** published on page 121 of this issue.

Discussion on Dr. Taylor's Notes.

Mr. C. A. Moore spoke of the effect of radium in this condition: the size of the spleen diminished very rapidly. Adrenalin has a very transient but well-marked effect in diminishing the bulk of the spleen. In operating, the adrenalin should be injected only when everything is ready for the actual removal. He commented on the good temperamental effect seen in this patient after splenectomy.

Dr. A. D. Fraser said that he considered this a case of Gaucher's disease in a malignant form.

The Medical Library of the University of Bristol,

WITH WHICH IS INCORPORATED

The Library of the Bristol Medico-Chirurgical Society.

The following donations have been received since the publication of the list in April, 1929.

	July, 1929.
University of Aberdeen (1)	1 volume
Dr. W. S. Bainbridge (2)	1 ,,
Dental Board of the United Kingdom (3) ..	1 ,,
Dr. A. L. Flemming (4)	1 ,,
Professor E. W. Hey Groves (5)	1 ,,
Henry Phipps Institute (6)	1 ,,
Government of India (7)	1 ,,
Dr. G. H. Jackson (8)	1 ,,
Dr. R. C. Leonard (9)	29 volumes
Monsieur A. Lumière (10)	1 volume
Michell Clarke Memorial Fund (11)	3 volumes
National Institute for Medical Research (12)..	1 pamphlet
Philadelphia General Hospital (13)	1 volume
Rockefeller Foundation (14)	1 ,,
Surgeon-General of the U.S. Army (15) ..	1 ,,

Unbound periodicals have also been received from Professor Hey Groves, Dr. Carey Coombs and Dr. C. Corfield.

THE ONE HUNDRED AND TWENTY-EIGHTH LIST OF BOOKS.

The figures in round brackets refer to the figures after the names of the donors. The books to which no such figures are attached have either been bought from the Library Fund or received through the *Journal*.

Alport, C. A.	*On Nephritis*		1929
Angle, E. H.	*Malocclusion of the Teeth*	7th Ed. (11)	1907
Barton, E. A.	*Essentials of Infant Feeding*	2nd Ed.	1929
Bennett, T. I.	*Nephritis: its Problems and Treatment*		1929
Bourne, G. and Stone, K.	*Principles of Clinical Pathology in Practice*		1929

Carrington, R. E.	*Notes on Pathology*		(9)	1892
Chagas, C.	*On the Practice of Terminal Disinfection*			1928
Giles, A. E.	*Gynæcological Diagnosis*		(9)	1906
Gladstone, H. B.	*Infant Feeding and Nutrition*			1928
Hamer, Sir W.	*Epidemiology, Old and New*			1928
Harrower, H. R.	*Practical Organotherapy*	4th Ed.	(9)	1922
Hertzler, A. E.	*Technic of Local Anæsthesia*	4th Ed.	(4)	1928
Hess, J. H.	*Feeding and the Nutritional Disorders of Infancy*	6th Ed.	(11)	1928
Hopewell-Ash, E.	*Therapy of Personal Influence*			1929
Jones, D. W. C.	*Elementary Medicine in terms of Physiology*			1929
Lewis, T.	*Blood Vessels of the Human Skin*		(11)	1927
Leys, D.	*Chronic Pulmonary Catarrh*			1927
	Local Analgesia in Dental Practice		(3)	1929
McCurrich, H. J.	*Treatment of the Sick Poor of this Country*			1929
MacWilliam, J. A., and others.	*Physiological Studies*	2nd Series	(1)	1926
	Practical Methods of Urine Analysis		(9)	1899
Saalfeld, E.	*Lectures on Cosmetic Treatment*		(9)	1911
Sauerbruch, F.	*Chirurgie der Brustorgane I*, i.		(5)	1928
Smith, F. J.	*Forensic Medicine and Toxicology*	3rd Ed.		1929
Whittaker, C. R.	*Manual of Surgical Anatomy*	2nd Ed.	(9)	1914

TRANSACTIONS, REPORTS, JOURNALS, ETC.

Methods and Problems of Medical Education. 12th Series	(14)	1929
Progressive Medicine. June, 1929		1929
Report of the Henry Phipps Institute	(6)	1928
Report of the International Congress on Military Medicine and Pharmacy. 1927	(2)	1929
Studies from the Laboratories of the Philadelphia General Hospital. v.	(13)	1928
Surgical Clinics of North America. Feb., 1929		1929
Transactions of the 7th Congress of the Far Eastern Association of Tropical Medicine. Vol. I.	(7)	1928

Local Medical Notes.

EXAMINATION RESULTS.

F.R.C.S.—Robert Victor Cooke, M.B., Ch.B., Bristol.

University of Bristol.—Students of the University have recently passed the following examinations :—

M.B., CH.B.—*First Examination. Pass :* Dorothy E. Barber, A. G. W. Branch, Rosalind M. S. Derham, Violet Fry,

A. V. House, A. D. Jones, C. H. G. Price, C. F. Rivington, S. Roberts, Mary G. Thomas, Marion L. Tryon.

M.B., CH.B.—*Final Examination* (*Part I.*). *Pass: In Forensic Medicine and Toxicology only*, A. J. B. Miall, H. M. Strover, W. L. Sleight.

M.B., CH.B.—*Second Examination* (*Part II.*). *Pass:* J. R. Gibbs, H. James.

M.B., CH.B.—*Final Examination* (*Part II.*). *Pass:* With Second Class Honours, R. A. S. Cory (with distinction in Pathology, Medicine and Obstetrics), R. J. Krogh (with distinction in Medicine and Obstetrics). *Pass:* Isabella J. Armstrong, A. C. Fisher, April D. James (with distinction in Public Health). *In Group II.* (*Completing Examination*): E. Southam. *In Group I.:* N. D. Gerrish, Mabel F. Potter. *In Group II.:* N. L. Price.

B.D.S.—*Third Examination. Pass: In Dental Metallurgy and Dental Mechanics* (*Completing Examination*), Muriel E. S. Cosh. *In Dental Materia Medica* (*Completing Examination*): W. Forsyth. *In Dental Metallurgy and Dental Mechanics only:* M. J. Ryan.

L.D.S.—*First Professional Examination. Pass: In Physiology and Anatomy* (*Completing Examination*), R. E. W. Smith. *In Physiology and Anatomy only:* Helena C. L. Blinkworth, G. N. Minifie, J. H. G. Smith.

L.D.S.—*Second Professional Examination. Pass: In Dental Materia Medica only*, E. V. O'Hara, H. L. Saunders.

L.D.S.—*Final Professional Examination. Pass:* C. R. H. Edwards, L. W. G. Jones.

CONJOINT BOARD.—M.R.C.S., L.R.C.P. (*Final Examination*), C. E. Brierley (in Surgery).

M.R.C.S., L.R.C.P.—R. J. Krogh, J. V. Lucas, K. H. Pridie, N. C. Tanner, G. J. S. West.

L.D.S., R.C.S.—*First Professional Examination* (*Part I.*). *Pass:* E. R. Dowland, T. A. A. Eaves, D. R. Jones, W. R. R. Mewton. *Part II. Pass:* G. H. Dakers, E. S. Edwards, W. U. Harwood, A. M. Watson, J. H. White. *Part III.* (*b*). *Pass:* E. R. Dowland, I. M. Ayliffe, W. A. Duly, R. E. Morgan.